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     (FILE 'HOME' ENTERED AT 13:42:14 ON 15 NOV 2001)

     FILE 'MEDLINE, AGRICOLA, CAPLUS, BIOSIS, EMBASE, WPIDS' ENTERED AT
     13:42:30 ON 15 NOV 2001
L1             6 S YAHN
L2             5 DUP REM L1 (1 DUPLICATE REMOVED)
L3           838 S LYSE AND (AMINO (W) ACID)
L4            46 S L3 AND TRANSMEMBRANE
L5            21 DUP REM L4 (25 DUPLICATES REMOVED)

     FILE 'CAPLUS' ENTERED AT 13:53:45 ON 15 NOV 2001

     FILE 'CAPLUS' ENTERED AT 13:53:58 ON 15 NOV 2001
                E LIVSHITS V A/AU 25
L6             1 S (E3 OR E18 OR E19 OR E20 OR E21) AND (YAHN)

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=> d 1- ibib abs
YOU HAVE REQUESTED DATA FROM 5 ANSWERS - CONTINUE? Y/(N):y

L2 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:456755 CAPLUS
DOCUMENT NUMBER: 133:85119
TITLE: Production of L-amino acids by bacterium transformed with amino acid excretion protein homologs
INVENTOR(S): Livshits, Vitaliy Arkadievich; Zakataeva, Natalia Pavlovna; Nakanishi, Kazuo; Aleshin, Vladimir Veniaminovich; Troshin, Petr Vladimirovich; Tokhmakova, Irina Lyvovna
PATENT ASSIGNEE(S): Ajinomoto Co., Inc., Japan
SOURCE: Eur. Pat. Appl., 29 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1016710	A2	20000705	EP 1999-125263	19991217
EP 1016710	A3	20000906		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
AU 9964493	A1	20000706	AU 1999-64493	19991213
JP 2000189180	A2	20000711	JP 1999-373651	19991228
BR 9906287	A	20010123	BR 1999-6287	19991228
CN 1261626	A	20000802	CN 1999-127522	19991230

PRIORITY APPLN. INFO.: RU 1998-124016 A 19981230
RU 1999-104431 A 19990309

AB A bacterium belonging to the genus Escherichia is provided having an ability to produce an L-amino acid, wherein the ability to produce the L-amino acid is increased by increasing an expression amt. of an L-amino acid excretion protein. Thus, genes **yahN**, yfiK, yeaS, and yggA are isolated by PCR amplification and shown to have homol. with lysine transporter LysE of Corynebacterium glutamicum and RhtB protein. When these genes are amplified in E. coli, the transformed organism shows increased levels of L-amino acid prodn.

L2 ANSWER 2 OF 5 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000427116 MEDLINE
DOCUMENT NUMBER: 20397473 PubMed ID: 10943564
TITLE: The LysE superfamily: topology of the lysine exporter LysE of Corynebacterium glutamicum, a paradyme for a novel superfamily of transmembrane solute translocators.
AUTHOR: Vrljic M; Garg J; Bellmann A; Wachi S; Freudl R; Malecki M J; Sahm H; Kozina V J; Eggeling L; Saier M H Jr; Eggeling L; Saier M H Jr
CORPORATE SOURCE: Institut fur Biotechnologie, Forschungszentrum Julich GmbH, Germany.
CONTRACT NUMBER: 5RO1 AI21702 (NIAID)
9RO1 GM55434 (NIGMS)
SOURCE: JOURNAL OF MOLECULAR MICROBIOLOGY AND BIOTECHNOLOGY, (1999 Nov) 1 (2) 327-36.
Journal code: DSF; 100892561. ISSN: 1464-1801.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200009
ENTRY DATE: Entered STN: 20000922
Last Updated on STN: 20000922
Entered Medline: 20000912

AB In Corynebacterium glutamicum the LysE carrier protein exhibits the unique function of exporting L-lysine. We here analyze the membrane topology of LysE, a protein of 236 amino acyl residues, using PhoA- and LacZ-fusions. The amino-terminal end of LysE is located in the cytoplasm whereas the carboxy-terminal end is found in the periplasm. Although 6 hydrophobic domains were identified based on hydropathy analyses, only five transmembrane spanning helices appear to be present. The additional hydrophobic segment may dip into the membrane or be surface localized. We show that LysE is a member of a family of proteins found, for example, in Escherichia coil, Bacillus subtilis, Mycobacterium tuberculosis and Helicobacter pylori. This family, which we have designated the LysE family, is distantly related to two additional protein families which we have designated the **YahN** and CadD families. These three families, the members of which exhibit similar sizes, hydropathy profiles, and sequence motifs comprise the LysE superfamily. Functionally characterized members of the LysE superfamily export L-lysine, cadmium and

possibly quarternary amines. We suggest that LysE superfamily members will prove to catalyze export of a variety of biologically important solutes.

L2 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1993:179008 CAPLUS
DOCUMENT NUMBER: 118:179008
TITLE: Electron spectroscopy of alkali-doped fullerene (C60)
AUTHOR(S): Takahashi, Takashi
CORPORATE SOURCE: Fac. Sci., Tohoku Univ., Sendai, 980, Japan
SOURCE: Nippon Butsuri Gakkaishi (1993), 48(1), 40-3
CODEN: NBGSAW; ISSN: 0369-3503
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A brief review with 11 refs. has been given of the recent studies on the electron spectroscopy of alkali metal-doped C6.0 superconductors. The photoemission and inverse photoemission spectra invalidate the applicability of the rigid band hypothesis and indicate the formation of a pseudo-gap, which owes probably to the on-site Coulomb interaction between C2p electrons and also to the lattice distortion induced by doped electrons through the **Yahn**-Teller effect. A comparison is made with the features of Cu2O3 compd. superconductors.

L2 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1982:554748 CAPLUS
DOCUMENT NUMBER: 97:154748
TITLE: Relation between dipole moments of symmetric metal tris-.beta.-diketonates and structure of the coordination unit
AUTHOR(S): Gerasimchuk, A. I.; Mazurenko, E. A.; Volkov, S. V.
CORPORATE SOURCE: Inst. Obshch. Neorg. Khim., Kiev, USSR
SOURCE: Ukr. Khim. Zh. (Russ. Ed.) (1982), 48(8), 787-90
CODEN: UKZHAU; ISSN: 0041-6045
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB The dipole moment of tris-.beta.-diketonates of metals can be explained by the asymmetry of the coordination unit, which shows a pseudo Jahn-Teller effect. Values for Al, Ga, and In complexes are considered.

L2 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1981:577833 CAPLUS
DOCUMENT NUMBER: 95:177833
TITLE: ESR of JTE (**Yahn**-Teller effect). III
AUTHOR(S): Mizuhashi, Seiji
CORPORATE SOURCE: Appl Sci. Lab., Electro-Commun. Univ., Tokyo, 182, Japan
SOURCE: Denki Tsushin Daigaku Gakuho (1981), 31(2), 257-61
CODEN: DTDRAU
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 8 refs.

=> s lyse and (amino (w) acid)
3 FILES SEARCHED...
L3 838 LYSE AND (AMINO (W) ACID)

=> s l3 and transmembrane
L4 46 L3 AND TRANSMEMBRANE

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5 21 DUP REM L4 (25 DUPLICATES REMOVED)

=> d 1- ibib abs
YOU HAVE REQUESTED DATA FROM 21 ANSWERS - CONTINUE? Y/(N):y

L5 ANSWER 1 OF 21 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001351785 MEDLINE
DOCUMENT NUMBER: 21308527 PubMed ID: 11414734
TITLE: A "chimeric" C57l-derived Ly49 inhibitory receptor resembling the Ly49D activation receptor.
AUTHOR: Mehta I K; Smith H R; Wang J; Margulies D H; Yokoyama W M
CORPORATE SOURCE: Immunology Program and Rheumatology Division, Washington University School of Medicine, St. Louis, Missouri 63110, USA.
CONTRACT NUMBER: 5T32AI07163 (NIAID)
SOURCE: CELLULAR IMMUNOLOGY, (2001 Apr 10) 209 (1) 29-41.
Journal code: CQ9; 1246405. ISSN: 0008-8749.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

OTHER SOURCE: GENBANK-AY003920
ENTRY MONTH: 200107
ENTRY DATE: Entered STN: 20010730
Last Updated on STN: 20010730
Entered Medline: 20010726

AB Ly49D is a natural killer (NK) cell activation receptor that is responsible for differential mouse inbred strain-determined lysis of Chinese hamster ovary (CHO) cells. Whereas C57BL/6 NK cells kill CHO, BALB/c-derived NK cells cannot kill because they lack expression of Ly49D. Furthermore, the expression of Ly49D, as detected by monoclonal antibody 4E4, correlates well with CHO lysis by NK cells from different inbred strains. However, one discordant mouse strain was identified; C57L NK cells express the mAb 4E4 epitope but fail to **lyse** CHO cells. Herein we describe a Ly49 molecule isolated from C57L mice that is recognized by mAb 4E4 (anti-Ly49D). Interestingly, this molecule shares extensive similarity to Ly49D(B6) in its extracellular domain, but its cytoplasmic and **transmembrane** domains are identical to the inhibitory receptor Ly49A(B6), including a cytoplasmic ITIM. This molecule bears substantial overall homology to the previously cloned Ly49O molecule from 129 mice the serologic reactivity and function of which were undefined. Cytotoxicity experiments revealed that 4E4(+) LAK cells from C57L mice failed to **lyse** CHO cells and inhibited NK cell function in redirected inhibition assays. MHC class I tetramer staining revealed that the Ly49O(C57L)-bound H-2D(d) and lysis by 4E4(+) C57L LAK cells is inhibited by target H-2D(d). The structural basis for ligand binding was also examined in the context of the recent crystallization of a Ly49A-H-2D(d) complex. Therefore, this apparently "chimeric" Ly49 molecule serologically resembles an NK cell activation receptor but functions as an inhibitory receptor. Copyright 2001 Academic Press.

L5 ANSWER 2 OF 21 MEDLINE
ACCESSION NUMBER: 1999436116 MEDLINE
DOCUMENT NUMBER: 99436116 PubMed ID: 10506166
TITLE: The interactions of histidine-containing amphipathic helical peptide antibiotics with lipid bilayers. The effects of charges and pH.
AUTHOR: Vogt T C; Bechinger B
CORPORATE SOURCE: Max-Planck-Institute for Biochemistry, Am Klopferspitz 18A, 82152 Martinsried, Germany.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Oct 8) 274 (41) 29115-21.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199911
ENTRY DATE: Entered STN: 20000111
Last Updated on STN: 20000111
Entered Medline: 19991109

AB The alpha-helix of the designed amphipathic peptide antibiotic LAH(4)(KKALLALALHHLAHLALHLALALKKA-NH(2)) strongly interacts with phospholipid membranes. The peptide is oriented parallel to the membrane surface under acidic conditions, but **transmembrane** at physiological pH (Bechinger, B. (1996) J. Mol. Biol. 263, 768-775). LAH(4) exhibits antibiotic activities against Escherichia coli and Bacillus subtilis; the peptide does not, however, **lyse** human red blood cells at bacteriocidal concentrations. The antibiotic activities of LAH(4) are 2 orders of magnitude more pronounced at pH 5 when compared with pH 7.5. Although peptide association at low pH is reduced when compared with pH 7.5, the release of the fluorophore calcein from large unilamellar 1-palmitoyl-2-oleoyl-sn-glycerol-3-phosphocholine or 1-palmitoyl-2-oleoyl-sn-glycero-3-phosphoglycerol vesicles is more pronounced at pH values where LAH(4) adopts an orientation along the membrane surface. The calcein release experiments thereby parallel the results obtained in antibiotic assays. Despite a much higher degree of association, calcein release activity of LAH(4) is significantly decreased for negatively charged membranes. Pronounced differences in the interactions of LAH(4) with 1-palmitoyl-2-oleoyl-sn-glycero-3-phosphoglycerol or 1-palmitoyl-2-oleoyl-sn-glycerol-3-phosphocholine membranes also become apparent when the mechanisms of dye release are investigated. The results presented in this paper support models in which antibiotic activity is caused by detergent-like membrane destabilization, rather than pore formation by helical peptides in **transmembrane** alignments.

L5 ANSWER 3 OF 21 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999115463 MEDLINE
DOCUMENT NUMBER: 99115463 PubMed ID: 9914515
TITLE: A comparative study on the structure and function of a cytolytic alpha-helical peptide and its antimicrobial beta-sheet diastereomer.
AUTHOR: Oren Z; Hong J; Shai Y

CORPORATE SOURCE: Department of Biological Chemistry, Weizmann Institute of Science, Rehovot, Isreal.
SOURCE: EUROPEAN JOURNAL OF BIOCHEMISTRY, (1999 Jan) 259 (1-2) 360-9.
Journal code: EMZ; 0107600. ISSN: 0014-2956.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199903
ENTRY DATE: Entered STN: 19990324
Last Updated on STN: 19990324
Entered Medline: 19990305

AB Antimicrobial peptides which adopt mainly or only beta-sheet structures have two or more disulfide bonds stabilizing their structure. The disruption of the disulfide bonds results in most cases in a large decrease in their antimicrobial activity. In the present study we examined the effect of d-amino acids incorporation on the structure and function of a cytolytic alpha-helical peptide which acts on erythrocytes and bacteria. The influence of a single or double d-**amino acid** replacement in alpha-helical peptides on their structure was reported previously in 50% 2,2,2, trifluoroethanol/water [Krause et al. (1995) Anal. Chem. 67, 252-258]. Here we used Attenuated Total Reflectance Fourier-Transform Infrared (ATR-FTIR) spectroscopy and found that the predominant structure of the wild-type peptide is alpha-helix in phospholipid membranes, whereas the structure of the diastereomer is beta-sheet. However, the linear, beta-sheet diastereomer preserved its cytolytic activity on bacteria but not on erythrocytes. Previous studies have shown that the ability of antimicrobial peptides to **lyse** bacteria but not normal mammalian cells correlated with their ability to disintegrate preferentially negatively charged, but not zwitterionic phospholipid membranes. In contrast, the diastereomer described here disrupts zwitterionic and negatively charged vesicles with similar potencies to those of the hemolytic wild-type peptide. Interestingly, whereas addition of a positive charge to the N-terminus of the wild-type peptide (which caused a minor effect on its structure) increased activity only towards some of the bacteria tested, similar modification in the diastereomer increased activity towards all of them. Furthermore, the modified wild-type peptide preserved its potency to destabilize zwitterionic and negatively charged vesicles, whereas the modified diastereomer had a reduced potency on zwitterionic vesicles but increased potency on negatively charged vesicles. Overall our results suggest that this new class of antimicrobial diastereomeric peptides bind to the membrane in 'carpet-like' manner followed by membrane disruption and breakdown, rather than forming a **transmembrane** pore which interfere with the bacteria potential. These studies also open a way to design new broad-spectrum antibacterial peptides.

L5 ANSWER 4 OF 21 MEDLINE
ACCESSION NUMBER: 2000427116 MEDLINE
DOCUMENT NUMBER: 20397473 PubMed ID: 10943564
TITLE: The **LysE** superfamily: topology of the lysine exporter **LysE** of Corynebacterium glutamicum, a paradyme for a novel superfamily of **transmembrane** solute translocators.
AUTHOR: Vrljic M; Garg J; Bellmann A; Wachi S; Freudl R; Malecki M J; Sahm H; Kozina V J; Eggeling L; Saier M H Jr; Eggeling L; Saier M H Jr
CORPORATE SOURCE: Institut fur Biotechnologie, Forschungszentrum Julich GmbH, Germany.
CONTRACT NUMBER: 5RO1 AI21702 (NIAID)
9RO1 GM55434 (NIGMS)
SOURCE: JOURNAL OF MOLECULAR MICROBIOLOGY AND BIOTECHNOLOGY, (1999 Nov) 1 (2) 327-36.
Journal code: DSF; 100892561. ISSN: 1464-1801.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200009
ENTRY DATE: Entered STN: 20000922
Last Updated on STN: 20000922
Entered Medline: 20000912

AB In Corynebacterium glutamicum the **LysE** carrier protein exhibits the unique function of exporting L-lysine. We here analyze the membrane topology of **LysE**, a protein of 236 amino acyl residues, using PhoA- and LacZ-fusions. The amino-terminal end of **LysE** is located in the cytoplasm whereas the carboxy-terminal end is found in the periplasm. Although 6 hydrophobic domains were identified based on hydropathy analyses, only five **transmembrane** spanning helices appear to be present. The additional hydrophobic segment may dip into the membrane or be surface localized. We show that **LysE** is a member

of a family of proteins found, for example, in Escherichia coil, Bacillus subtilis, Mycobacterium tuberculosis and Helicobacter pylori. This family, which we have designated the **LysE** family, is distantly related to two additional protein families which we have designated the YahN and CadD families. These three families, the members of which exhibit similar sizes, hydropathy profiles, and sequence motifs comprise the **LysE** superfamily. Functionally characterized members of the **LysE** superfamily export L-lysine, cadmium and possibly quarternary amines. We suggest that **LysE** superfamily members will prove to catalyze export of a variety of biologically important solutes.

L5 ANSWER 5 OF 21 MEDLINE
ACCESSION NUMBER: 1998266202 MEDLINE
DOCUMENT NUMBER: 98266202 PubMed ID: 9605166
TITLE: Potassium leakage during the apoptotic degradation phase.
AUTHOR: Dallaporta B; Hirsch T; Susin S A; Zamzami N; Larochette N; Brenner C; Marzo I; Kroemer G
CORPORATE SOURCE: Centre National de Recherche Scientifique, Villejuif, France.
SOURCE: JOURNAL OF IMMUNOLOGY, (1998 Jun 1) 160 (11) 5605-15. Journal code: IFB; 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980618
Last Updated on STN: 20000303
Entered Medline: 19980611

AB The subcellular compartmentalization of ions is perturbed during the process of apoptosis. In this work, we investigated the impact of K+ on the apoptotic process in thymocytes and T cell hybridoma cells. Irrespective of the death-inducing stimulus (glucocorticoids, topoisomerase inhibition, or Fas-crosslinking), a significant K+ outflow was observed during apoptosis, as determined on the single-cell level by means of the K+-sensitive fluorochrome, benzofuran isophtalate. This loss of cytosolic K+ only occurs in cells that have completely disrupted their inner mitochondrial **transmembrane** potential. Inhibition of this mitochondrial **transmembrane** potential loss by Bcl-2 or by specific inhibitors acting on the mitochondrial permeability transition pore (bongkrekic acid, cyclosporin A) prevents K+ leakage. K+ drops at the same stage at which cells expose phosphatidylserine residues on the outer leaflet of the membrane and reduce the levels of nonoxidized glutathione, but before they hyperproduce reactive oxygen species, undergo massive Ca2+ influx, shrink, and **lyse**. In a cell-free system of apoptosis, isolated nuclei exposed to the supernatant of mitochondria that have undergone permeability transition only manifest chromatinolysis when the K+ concentration is lowered from physiologic to apoptotic levels. Accordingly, massive DNA fragmentation causing subdiploidy is confined to cells that have undergone K+ leakage. Together, these data point to the step-wise acquisition of membrane dysfunction in apoptosis and indicate an important role for the disruption of normal K+ homeostasis in apoptotic degradation. Derepression of endonucleases due to low K+ concentrations may be a decisive prerequisite for end-stage DNA fragmentation.

L5 ANSWER 6 OF 21 MEDLINE
ACCESSION NUMBER: 97444336 MEDLINE
DOCUMENT NUMBER: 97444336 PubMed ID: 9298965
TITLE: The concentration-dependent membrane activity of cecropin A.
COMMENT: Erratum in: Biochemistry 1999 Mar 23;38(12):3850
AUTHOR: Silvestro L; Gupta K; Weiser J N; Axelsen P H
CORPORATE SOURCE: Department of Pharmacology, Infectious Diseases Section, and Johnson Foundation for Molecular Biophysics, University of Pennsylvania, Philadelphia, Pennsylvania 19104-6084, USA.
CONTRACT NUMBER: GM50805 (NIGMS)
GM54617 (NIGMS)
HL47469 (NHLBI)
+
SOURCE: BIOCHEMISTRY, (1997 Sep 23) 36 (38) 11452-60. Journal code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 19971024
Last Updated on STN: 20000303
Entered Medline: 19971016

AB Cecropin A is a naturally occurring, linear, cationic, 37-residue antimicrobial peptide. The precise mechanism by which it kills bacteria is

not known, but its site of action is believed to be the cell membrane. To investigate the nature of its membrane activity, we examined the ability of cecropin A to alter membrane permeability in synthetic lipid vesicles and in Gram-negative bacteria. Cecropin A exerted distinctly different types of membrane activity depending on its concentration. In synthetic lipid vesicles, cecropin A dissipated **transmembrane** electrochemical ion gradients at relatively low concentrations, but much higher concentrations were required to release an encapsulated fluorescent probe. Cecropin A dissipated ion gradients whether or not the vesicle membranes contained anionic lipid, although the presence of anionic lipid dramatically increased peptide binding, and modestly increased the release of an encapsulated probe. Cholesterol did not prevent the dissipation of ion gradients by low concentrations of peptide, but it did inhibit release of the encapsulated probe by high concentrations of peptide. At the highest concentrations examined, cecropin A remained monomeric in solution, and did not aggregate, **lyse**, or otherwise alter vesicle size. In Gram-negative bacteria, cecropin A was potently bactericidal at concentrations which dissipated ion gradients in lipid vesicles, but much higher concentrations were required to cause the release of cytoplasmic contents. These findings point to the conclusion that cecropin A kills bacteria by dissipating **transmembrane** electrochemical ion gradients. They weigh against theories comparing the antimicrobial activity of cecropin A to the release of encapsulated probes from lipid vesicles, and against roles for cholesterol or anionic lipid headgroups in the selectivity of peptide action against bacteria.

L5 ANSWER 7 OF 21 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1998062194 MEDLINE
DOCUMENT NUMBER: 98062194 PubMed ID: 9401022
TITLE: cps1+, a Schizosaccharomyces pombe gene homolog of Saccharomyces cerevisiae FKS genes whose mutation confers hypersensitivity to cyclosporin A and papulacandin B.
AUTHOR: Ishiguro J; Saitou A; Duran A; Ribas J C
CORPORATE SOURCE: Department of Biology, Faculty of Science, Konan University, Okamoto, Kobe, Japan.
SOURCE: JOURNAL OF BACTERIOLOGY, (1997 Dec) 179 (24) 7653-62. Journal code: HH3; 2985120R. ISSN: 0021-9193.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-D78352
ENTRY MONTH: 199801
ENTRY DATE: Entered STN: 19980129
Last Updated on STN: 19980129
Entered Medline: 19980113

AB The Schizosaccharomyces pombe cps1-12 (for chlorpropham supersensitive) mutant strain was originally isolated as hypersensitive to the spindle poison isopropyl N-3-chlorophenyl carbamate (chlorpropham) (J. Ishiguro and Y. Uhara, Jpn. J. Genet. 67:97-109, 1992). We have found that the cps1-12 mutation also confers (i) hypersensitivity to the immunosuppressant cyclosporin A (CsA), (ii) hypersensitivity to the drug papulacandin B, which specifically inhibits 1,3-beta-D-glucan synthesis both in vivo and in vitro, and (iii) thermosensitive growth at 37 degrees C. Under any of these restrictive treatments, cells swell up and finally **lyse**. With an osmotic stabilizer, cells do not **lyse**, but at 37 degrees C they become multiseptated and multibranched. The cps1-12 mutant, grown at a restrictive temperature, showed an increase in sensitivity to lysis by enzymatic cell wall degradation, in in vitro 1,3-beta-D-glucan synthase activity (173% in the absence of GTP in the reaction), and in cell wall biosynthesis (130% of the wild-type amount). Addition of Ca2+ suppresses hypersensitivity to papulacandin B and septation and branching phenotypes. All of these data suggest a relationship between the cps1+ gene and cell wall synthesis. A DNA fragment containing the cps1+ gene was cloned, and sequence analysis indicated that it encodes a predicted membrane protein of 1,729 amino acids with 15 to 16 **transmembrane** domains. S. pombe cps1p has overall 55% sequence identity with Fks1p or Fks2p, proposed to be catalytic or associated subunits of Saccharomyces cerevisiae 1,3-beta-D-glucan synthase. Thus, the cps1+ product might be a catalytic or an associated copurifying subunit of the fission yeast 1,3-beta-D-glucan synthase that plays an essential role in cell wall synthesis.

L5 ANSWER 8 OF 21 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1997:332314 CAPLUS
DOCUMENT NUMBER: 127:134455
TITLE: The HER2/neu-derived peptide p654-662 is a tumor-associated antigen in human pancreatic cancer recognized by cytotoxic T lymphocytes
AUTHOR(S): Peiper, Matthias; Goedegebuure, Peter S.; Linehan, David C.; Ganguly, Eric; Douville, Cara C.; Eberlein,

Timothy J.
CORPORATE SOURCE: Brigham Women's Hospital, Harvard Medical School, Boston, MA, 02115, USA
SOURCE: Eur. J. Immunol. (1997), 27(5), 1115-1123
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: VCH
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The protooncogene HER2/neu encodes a 185-kDa **transmembrane** protein with extensive homol. to the epidermal growth factor receptor. It is overexpressed in several human cancers of epithelial origin, such as pancreatic cancer. Cytotoxic T lymphocytes (CTL) derived from breast, ovarian, and non-small cell lung cancer recognized a peptide derived from HER2/neu. To evaluate whether this HLA-A2-binding peptide is a tumor-assocd. antigen (TAA) in pancreatic cancer, the ability of HER2/neu-reactive CTL was tested to **lyse** human pancreatic carcinoma cells. CTL were generated from tumor-assocd. T lymphocytes from HLA-A2+HER2/neu+ breast and ovarian cancer patients. All CTL recognized autologous and allogeneic HER2/ neu+ tumor cells in an HLA-A2-restricted fashion. All CTL recognized p654-662 (GP2) derived from HER2/neu. These CTL also recognized HER2/neu+ pancreatic cancer cells in an HLA-A2-restricted fashion. HER2/neu+ HLA-A2- pancreatic cancer cells were not or only poorly lysed. Repeated stimulation of HLA-A2+ PBL from pancreatic cancer patients using the HER2/neu-derived peptide resulted in specific recognition of this peptide and, more importantly, HER2/neu+ pancreatic tumors in an HLA-A2-restricted fashion. Autologous HLA-A2+ fibroblasts or HLA-A2+ malignant melanoma cells were not recognized. HLA-A2- peptide-stimulated T lymphocytes showed no cytotoxicity. These results demonstrate that this HER2/neu-derived peptide is a shared TAA among several adenocarcinomas including pancreatic carcinoma, suggesting a common mechanism of recognition of these human tumors by T lymphocytes. The identification of the HER2/neu-derived peptide GP2 as a TAA in pancreatic cancer provides an opportunity for the design of novel immunotherapy and vaccine strategies.

L5 ANSWER 9 OF 21 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1996:345795 CAPLUS
DOCUMENT NUMBER: 125:26231
TITLE: Cells bearing CD4 extracellular domain fusion products as decoys for the killing of HIV-1-infected cells
INVENTOR(S): Seed, Brian; Banapour, Babak; Romeo, Charles; Kolanus, Waldemar
PATENT ASSIGNEE(S): General Hospital Corporation, USA
SOURCE: PCT Int. Appl., 133 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9603883	A1	19960215	WO 1995-US9468	19950726
W: AU, BR, BY, CA, CN, CZ, FI, HU, JP, KR, MX, NO, NZ, PL, RU, SI, UA				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5851828	A	19981222	US 1994-284391	19940802
AU 9532014	A1	19960304	AU 1995-32014	19950726
AU 697489	B2	19981008		
EP 781095	A1	19970702	EP 1995-928152	19950726
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, PT, SE				
JP 10503932	T2	19980414	JP 1995-506600	19950726
PL 181085	B1	20010531	PL 1995-318443	19950726
FI 9700428	A	19970326	FI 1997-428	19970131
NO 9700440	A	19970326	NO 1997-440	19970131

PRIORITY APPLN. INFO.:

US 1994-284391	A	19940802
US 1995-394388	A	19950224
US 1991-665961	B2	19910307
US 1992-847566	B2	19920306
US 1994-195395	B2	19940214
WO 1995-US9468	W	19950726

AB Cells that carry a surface mol. that has the extracellular domain of the CD4 antigen fused to a **transmembrane** receptor are described for identification and killing of HIV-1-infected cells in the treatment of HIV-1 infection. The chimeric receptor is presented by a cell capable of killing bound cells, e.g. a cytotoxic T-lymphocyte or a natural killer cell, and the binding of the antigen activates the intracellular domain of the receptor that activates the cell to kill the infected cell. The CD4 antigen domain is linked to the intracellular domain of a receptor such as a T-cell receptor, a B-cell receptor, or an Fc receptor, preferably by an Ig hinge and CH2 and CH3 domains to ensure correct spacing of the domains. Cells that express these CD4 receptors and DNA and vectors encoding the receptors are described. Fusion proteins with the intracellular domains

of the .zeta.-, .gamma.- and .eta.-chains of the T-cell receptor were synthesized in animal cell hosts where they were able to assoc. with the Fc.gamma.RIII receptor. These cells were able **lyse** cells presenting a gp120/gp41 complex.

L5 ANSWER 10 OF 21 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 96330322 MEDLINE
DOCUMENT NUMBER: 96330322 PubMed ID: 8760284
TITLE: Horse complement protein C9: primary structure and cytotoxic activity.
AUTHOR: Esser A F; Tarnuzzer R W; Tomlinson S; Tatar L D; Stanley K K
CORPORATE SOURCE: Department of Comparative and Experimental Pathology, University of Florida Health Science Center, Gainesville, USA.
CONTRACT NUMBER: F06 TW01402 (FIC)
R01 AI-19478 (NIAID)
SOURCE: MOLECULAR IMMUNOLOGY, (1996 May-Jun) 33 (7-8) 725-33.
Journal code: NG1; 7905289. ISSN: 0161-5890.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U19381
ENTRY MONTH: 199609
ENTRY DATE: Entered STN: 19961008
Last Updated on STN: 19961008
Entered Medline: 19960924

AB Lack of hemolytic activity of horse serum is an inherent property of horse C9. To understand the molecular reasons for this deficiency we have cloned C9 cDNA from a horse liver cDNA library and have sequenced the cDNA yielding the complete coding sequence for horse C9. Purification of C9 from horse plasma and microsequencing established the N-terminus of the mature protein and verified that the correct horse C9 cDNA clone had been isolated. The deduced **amino acid** sequence corresponds to a mature protein of 526 amino acids that is 77% identical to human C9. It has the same domain structure as human C9 and contains 22 cysteines and four invariant tryptophans. The few differences include the N-terminus, which is an unblocked glycine in horse C9 but pyroglutamine in human C9, and three potential N-glycosylation sites compared to two in human C9. The N-terminal difference is unimportant since microsequencing of bovine C9, which is strongly hemolytic, established that it also has an unblocked glycine identical to horse C9. There are no obvious structural differences apparent that could resolve the differences in hemolytic potency between the two molecules. Aside from a few conservative replacements, both C9 sequences are identical between positions 250 and 360. This region includes the membrane interaction domain in C9 and the postulated **transmembrane** segment that is thought to constitute the wall of a putative **transmembrane** pore and, therefore, should be required for cytotoxicity. In agreement with this prediction we have observed that, in contrast to the marked decrease in hemolytic activity, horse C9 is very efficient in killing a variety of Gram-negative bacteria. These results demonstrate that horse C9 is a structurally competent molecule with efficient cytotoxic activity. Its inability to **lyse** erythrocytes may be related to the action of control proteins on target cell membranes.

L5 ANSWER 11 OF 21 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 96343861 MEDLINE
DOCUMENT NUMBER: 96343861 PubMed ID: 8760804
TITLE: The natural killer cell receptor specific for HLA-A allotypes: a novel member of the p58/p70 family of inhibitory receptors that is characterized by three immunoglobulin-like domains and is expressed as a 140-kD disulphide-linked dimer.
AUTHOR: Pende D; Biassoni R; Cantoni C; Verdiani S; Falco M; di Donato C; Accame L; Bottino C; Moretta A; Moretta L
CORPORATE SOURCE: Istituto Scientifico Tumori, Genova, Italy.
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1996 Aug 1) 184 (2) 505-18.
Journal code: I2V; 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-X93595; GENBANK-X93596; GENBANK-X94262; GENBANK-X94373; GENBANK-X94374
ENTRY MONTH: 199609
ENTRY DATE: Entered STN: 19961008
Last Updated on STN: 19980206
Entered Medline: 19960923

AB Human natural killer (NK) cells express inhibitory receptors that are specific for different groups of HLA-C or HLA-B alleles. The majority of

these receptors belong to the immunoglobulin (Ig) superfamily and are characterized by two or three extracellular Ig-like domains. Here we describe a novel inhibitory NK receptor that is specific for a group of HLA-A alleles. The HLA-A3-specific NK cell clone DP7 has been used for mice immunization. Two mAbs, termed Q66 and Q241, bound to the immunizing clone and stained only a subset of NK cell populations or clones. Among Q66 mAb-reactive clones, we further selected those that did not express any of the previously identified HLA-class I-specific NK receptors. These clones did not **lyse** HLA-A3+ (or -A11+) target cells, but lysis of these targets could be detected in the presence of Q66 or Q241 mAbs. On the other hand, target cells expressing other HLA-A alleles, including -A1, -A2, and -A24, were efficiently lysed. Moreover, none of the HLA-C or HLA-B alleles that were tested exerted a protective effect. Q66+, but not Q66- NK cell clones, expressed messenger RNA coding for a novel 3 Ig domain protein homologous to the HLA-C (p58) and HLA-B (p70) receptors. The corresponding cDNA (cl.1.1) was used to generate transient and stable transfectants in COS7 and NIH3T3 cell lines, respectively. Both types of transfectants were specifically stained by Q66 and Q241 mAbs. Since the cytoplasmic tail of Q66-reactive molecules was at least 11 **amino acid** longer than the other known p58/p70 molecules, we could generate an antiserum specific for the COOH-terminus of Q66-reactive molecules, termed PGP-3. PGP-3 immunoprecipitated, only from Q66+ NK cells, molecules displaying a molecular mass of 140 kD, under nonreducing conditions, which resolved, under reducing conditions, in a 70-kD band. Thus, differently from the other p58/p70 receptors, Q66-reactive molecules appear to be expressed as disulphide-linked dimers and were thus termed p140. The comparative analysis of the **amino acid** sequences of p58, p70, and p140 molecules revealed the existence of two cysteins proximal to the **transmembrane** region, only in the **amino acid** sequence of p140 molecules.

L5 ANSWER 12 OF 21 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1996:46681 CAPLUS
DOCUMENT NUMBER: 124:76508
TITLE: Bifunctional, tumor antigen-binding proteins, cytotoxic T-cells expressing this protein, and their use in cancer treatment
INVENTOR(S): Groner, Bernd; Moritz, Dirk
PATENT ASSIGNEE(S): Ciba-Geigy A.-G., Switz.
SOURCE: PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9530014	A1	19951109	WO 1995-EP1494	19950420
W: AM, AU, BB, BG, BR, BY, CA, CN, CZ, EE, FI, GE, HU, IS, JP, KG, KP, KR, KZ, LK, LR, LT, LV, MD, MG, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, TJ, TM, TT, UA, US, UZ, VN				
RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2188422	AA	19951109	CA 1995-2188422	19950420
AU 9524469	A1	19951129	AU 1995-24469	19950420
AU 694222	B2	19980716		
EP 758394	A1	19970219	EP 1995-918576	19950420
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 09512176	T2	19971209	JP 1995-527965	19950420
TW 428026	B	20010401	TW 1995-84103999	19950424
ZA 9503440	A	19951102	ZA 1995-3440	19950428
FI 9604311	A	19961025	FI 1996-4311	19961025
NO 9604641	A	19961101	NO 1996-4641	19961101

PRIORITY APPLN. INFO.: EP 1994-810244 A 19940502
WO 1995-EP1494 W 19950420

AB The present invention relates to bifunctional protein capable of directing a host cell producing said protein to specifically recognize selected target cells. Furthermore, the invention provides a method for the prepn. of said protein, a DNA construct encoding said protein, a compn. comprising a host cell expressing said DNA, and antibodies specifically recognizing said protein. Addnl., the invention relates to the use of such a host cell, e.g. for selectively killing tumor cells in vitro and in vivo. A retroviral expression vector encoding a fusion protein consisting of a leader sequence from an Ig heavy chain, a single-chain Ig Fv fragment specific for extracellular domain of the erbB-2 receptor, a linker sequence from the Ig-like hinge region of CD8.alpha., and the **transmembrane** and signalling domain of the .zeta. chain of the T-cell receptor was prepd. Cytotoxic T-cells infected with this vector produced functional fusion protein and were able to efficiently **lyse** erbB-2 expressing cell lines in culture. Addnl., the

recombinant cytotoxic T-cells suppressed tumor formation in vivo.

L5 ANSWER 13 OF 21 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 96032743 MEDLINE
DOCUMENT NUMBER: 96032743 PubMed ID: 7559448
TITLE: Interactions between residues in staphylococcal alpha-hemolysin revealed by reversion mutagenesis.
AUTHOR: Panchal R G; Bayley H
CORPORATE SOURCE: Worcester Foundation for Biomedical Research, Shrewsbury, Massachusetts 01545, USA.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Sep 29) 270 (39) 23072-6.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199511
ENTRY DATE: Entered STN: 19951227
Last Updated on STN: 19951227
Entered Medline: 19951106

AB alpha-Hemolysin (alpha HL), a pore-forming polypeptide of 293 amino acids, is secreted by Staphylococcus aureus as a water-soluble monomer. Residues that play key roles in the formation of functional heptameric pores on rabbit red blood cells (rRBC) have been identified previously by site-directed mutagenesis. alpha HL-H35N, in which the histidine at position 35 of the wild-type sequence is replaced with asparagine, is nonlytic and is arrested in assembly as a heptameric prepore. In this study, second-site revertants of H35N that have the ability to **lyse** rRBC were generated by error-prone PCR under conditions designed to produce single base changes. The analysis of 22 revertants revealed new codons clustered predominantly in three distinct regions of the H35N gene. One cluster includes amino acids 107-111 (four revertants) and another residues 144-155 (five revertants). These two clusters flank the central glycine-rich loop of alpha HL, which previously has been implicated in formation of the **transmembrane** channel, and encompass residues Lys-110 and Asp-152 that, like His-35, are crucial for lytic activity. The third cluster lies in the region spanning amino acids 217-228 (eight revertants), a region previously unexplored by mutagenesis. Single revertants were found at **amino acid** positions 84 and 169. When compared with H35N, the heptameric prepores formed by the revertants underwent more rapid conversion to fully assembled pores, as determined by conformational analysis by limited proteolysis. The rate of conversion to the fully assembled pore was strongly correlated with hemolytic activity. Previous work has suggested that the N terminus of alpha HL and the central loop cooperate in the final step of assembly. The present study suggests that the key N-terminal residue His-35 operates in conjunction with residues flanking the loop and C-terminal residues in the region 217-228. Hence, reversion mutagenesis extends the linear analysis that has been provided by direct point mutagenesis.

L5 ANSWER 14 OF 21 MEDLINE
ACCESSION NUMBER: 95186500 MEDLINE
DOCUMENT NUMBER: 95186500 PubMed ID: 7533538
TITLE: Molecular basis for membrane selectivity of an antimicrobial peptide, magainin 2.
AUTHOR: Matsuzaki K; Sugishita K; Fujii N; Miyajima K
CORPORATE SOURCE: Faculty of Pharmaceutical Sciences, Kyoto University, Japan.
SOURCE: BIOCHEMISTRY, (1995 Mar 14) 34 (10) 3423-9.
Journal code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199504
ENTRY DATE: Entered STN: 19950425
Last Updated on STN: 19960129
Entered Medline: 19950413

AB Magainin peptides, isolated from Xenopus skin, kill bacteria by permeabilizing their cell membranes whereas they do not **lyse** erythrocytes. To elucidate the rationale for this membrane selectivity, we compared the effects of the membrane lipid composition and the **transmembrane** potential on the membrane-lytic power of magainin 2 with that of hemolytic melittin. The activity of magainin to zwitterionic phospholipids constituting the erythrocyte surface was extremely weak compared with that of melittin, and acidic phospholipids are necessary for effective action. The presence of sterols reduced the susceptibility of the membrane to magainin. The generation of an inside-negative **transmembrane** potential enhanced magainin-induced hemolysis. We can conclude that the absence of any acidic phospholipids on the outer monolayer and the abundant presence of cholesterol, combined with the lack

of the **transmembrane** potential, contribute to the protection of erythrocytes from magainin's attack.

L5 ANSWER 15 OF 21 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 95355829 MEDLINE
DOCUMENT NUMBER: 95355829 PubMed ID: 7629495
TITLE: Cloning and functional characteristics of murine large granular lymphocyte-1: a member of the Ly-49 gene family (Ly-49G2).
COMMENT: Comment in: J Exp Med. 1995 Aug 1;182(2):273-7
AUTHOR: Mason L H; Ortaldo J R; Young H A; Kumar V; Bennett M; Anderson S K
CORPORATE SOURCE: Laboratory of Experimental Immunology, PRI/DynCorp, NCI-FCRDC, Maryland 21702-1201, USA.
CONTRACT NUMBER: NO1-CO-74101 (NCI)
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1995 Aug 1) 182 (2) 293-303.
Journal code: I2V; 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199509
ENTRY DATE: Entered STN: 19950921
Last Updated on STN: 19950921
Entered Medline: 19950907

AB Large granular lymphocyte (LGL) 1 is a cell surface glycoprotein expressed on a subset (50%) of C57BL/6 natural killer (NK) cells. Immunoprecipitation experiments reveal that the LGL-1 protein exists as a disulfide-linked 40-kD homodimer. Functional studies of LGL-1+ cells indicate that selected H-2d target cells are not lysed efficiently by these interleukin (IL)-2-cultured NK cells. These findings suggested that LGL-1 may be a member of the Ly-49 gene family. Here we report the molecular cloning of the LGL-1 cDNA from a severe combined immunodeficient-adherent lymphokine-activated killer cell library transfected into Cos-7 cells and find LGL-1 to be homologous to the Ly-49 gene at both the nucleotide (85%) and **amino acid** levels (73%). Sequencing of our LGL-1 cDNA has revealed it to be nearly identical to the Ly-49G2 cDNA recently isolated by cross-hybridization with an Ly-49 probe. LGL-1 represents a type II **transmembrane** protein of 267 amino acids with its carboxyl end exposed extracellularly. The LGL-1 protein contains 11 highly conserved cysteine residues and a 25-**amino acid transmembrane** region. Southern blot analysis demonstrates that there are a number of homologous genes in mouse DNA that hybridize strongly to LGL-1. Northern analyses using poly A+ RNA from LGL-1+ NK cells indicate that LGL-1 is expressed as a 1.4 kb mRNA. Two-color flow cytometry analysis (FCA) of C57BL/6 splenic NK cells demonstrates that LGL-1 and Ly-49 label overlapping subsets of cells. FCA identifies four subsets of NK cells as defined by LGL-1 versus Ly-49 staining. We have sorted these individual subsets, expanded them in IL-2, and performed cytotoxicity experiments to determine their target cell profiles in relation to class I expression. Results of these studies are complex, but indicate that Ly-49 may not be the only molecule that recognizes class I as an inhibitory signal for cytotoxicity. LGL-1+ cells also fail to **lyse** several H-2d-expressing tumor targets and concanavalin A lymphoblasts from BALB/c but not C57BL/6 mice. This inhibition of lysis by LGL-1+ NK cells is negated by addition of monoclonal antibody (mAb) 4D11 that recognizes the LGL-1 protein. When mAbs to the class I molecules H-2Dd and H-2Ld (alpha 1 alpha 2 domains only) are added to cytotoxicity assays, LGL-1+ cells **lyse** H-2d targets very effectively.(ABSTRACT TRUNCATED AT 250 WORDS)

L5 ANSWER 16 OF 21 MEDLINE
ACCESSION NUMBER: 95375145 MEDLINE
DOCUMENT NUMBER: 95375145 PubMed ID: 7647218
TITLE: Tissue factor antigen levels in various biological fluids.
AUTHOR: Fareed J; Callas D D; Hoppensteadt D; Bermes E W Jr
CORPORATE SOURCE: Department of Pathology, Loyola University Medical Center, Maywood, IL 60153, USA.
SOURCE: BLOOD COAGULATION AND FIBRINOLYSIS, (1995 Jun) 6 Suppl 1 S32-6.
Journal code: A5J; 9102551. ISSN: 0957-5235.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199509
ENTRY DATE: Entered STN: 19951005
Last Updated on STN: 19951005
Entered Medline: 19950928

AB Tissue factor (TF), a **transmembrane** surface protein, is known to initiate thrombogenesis through plasmatic and cellular activation

processes. Besides complexing with factor VII, eventually leading to fibrin generation via the extrinsic pathway, TF can also activate factor IX, resulting in the intrinsic activation of coagulation. Other functions of TF are currently unknown, although various cells are believed to have TF receptors. Many of the post-surgical and post-interventional thrombotic events are due to the release of TF. Increased levels of TF are associated with several pathologic conditions such as cancer, sepsis and inflammation. Cellular necrosis also results in an increase of TF as the cells in the traumatized area **lyse** and release endogenous cell surface-bound TF. An ELISA method (American Diagnostica, Greenwich, CT) has been developed to assay TF antigen levels in various biological fluids. This ELISA employs a murine monoclonal antibody raised against native human TF for antigen capture. In this study, cerebrospinal fluid, peritoneal fluid, pleural effusion and urine from patients were assayed for their TF content using this ELISA method. Normal individual serum and plasma were also assayed as controls against which the levels of TF in the patients' body fluids could be compared. The amount of TF antigen in normal human plasma and serum was 165 +/- 139 pg/ml and 165 +/- 110 pg/ml, respectively. Concentrations of TF antigen in other fluids were: cerebrospinal fluid 868 +/- 721 pg/ml, peritoneal fluid 124 +/- 247 pg/ml, pleural effusion 385 +/- 569 pg/ml, synovial fluid 97 +/- 23 pg/ml, seminal plasma 11,485 +/- 875 pg/ml and urine 86 +/- 57 pg/ml.(ABSTRACT TRUNCATED AT 250 WORDS)

L5 ANSWER 17 OF 21 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 95151135 MEDLINE
DOCUMENT NUMBER: 95151135 PubMed ID: 7848510
TITLE: Major and minor Kb-restricted epitopes encoded by the endogenous ecotropic murine leukemia virus AKR623 that are recognized by anti-AKR/Gross MuLV CTL.
AUTHOR: White H D; Roeder D A; Lam T; Green W R
CORPORATE SOURCE: Department of Microbiology, Dartmouth Medical School, Lebanon, New Hampshire.
CONTRACT NUMBER: CA23108 (NCI)
CA36860 (NCI)
CA45049 (NCI)
SOURCE: VIRAL IMMUNOLOGY, (1994) 7 (2) 51-9.
Journal code: AD0; 8801552. ISSN: 0882-8245.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199503
ENTRY DATE: Entered STN: 19950322
Last Updated on STN: 19970203
Entered Medline: 19950316

AB C57BL/6 mice can generate a type-specific and class IH-2Kb-restricted CTL response against histocompatible AKR/Gross murine leukemia virus (MuLV) cell surface antigen positive (GCSA+) tumor cells. These anti-AKR/Gross MuLV CTL are also known to **lyse** SC.Kb/623 target cells expressing the molecular MuLV clone AKR623 (derived from the endogenous ecotropic MuLV provirus emv-11). To help identify AKR623 viral epitopes recognized by these CTL, four chimeric proviruses were constructed from two parental plasmids, pAKR623 and pAK7. It has been shown that SC.Kb/7 fibroblast targets expressing the emv-14-derived molecular clone AK7 are only poorly lysed by anti-AKR/Gross MuLV CTL. Data from experiments employing SC.Kb cells infected with the chimeras as targets against anti-AKR/Gross MuLV CTL supported the location of a previously identified immunodominant epitope located within the viral p15E **transmembrane** envelope protein, peptide TM134-141 (KSP-WFTTL). Furthermore, the use of Kb-motif-defined AKR623 encoded peptides together with data obtained using the chimeric viruses allowed the identification of three additional anti-AKR/Gross MuLV CTL epitopes. Peptides representing these epitopes, MA125-132 (RSALY-PAL), RT142-149 (SHRWYTVL), and RT456-463 (RMTHYQAM), are characterized herein with respect to their ability to confer lysis upon EMV- target cells and to stimulate tumor primed splenocytes in vitro. The identification and characterization of these additional epitopes allow for a better understanding of both the CTL response against GCSA+ tumor cells and the dysfunctional CTL response against EMV-14 and AK7.

L5 ANSWER 18 OF 21 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 93186847 MEDLINE
DOCUMENT NUMBER: 93186847 PubMed ID: 8444902
TITLE: Functional complementation of staphylococcal alpha-hemolysin fragments. Overlaps, nicks, and gaps in the glycine-rich loop.
AUTHOR: Walker B; Krishnasastry M; Bayley H
CORPORATE SOURCE: Worcester Foundation for Experimental Biology, Shrewsbury, MA 01545.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1993 Mar 5) 268 (7) 5285-92.
Journal code: HIV; 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199304
ENTRY DATE: Entered STN: 19930416
Last Updated on STN: 19930416
Entered Medline: 19930406

AB The final steps in assembly of the lytic pore formed by staphylococcal alpha-hemolysin (alpha HL) involve the formation of a nonlytic oligomeric pore precursor, followed by the formation of a **transmembrane** channel. In this study, truncation mutants of alpha HL encompassing the NH2-terminal or COOH-terminal half of the polypeptide chain and all, part, or none of the central glycine-rich loop were obtained by in vitro, coupled transcription and translation of mutant plasmid DNAs. These polypeptides were unable to oligomerize upon or cause lysis of rabbit erythrocytes (rRBCs). Twenty-one pairs of the same truncation mutants constituting discontinuous alpha HL chains with overlaps, nicks, and gaps in the central loop were obtained by cotranslation. When incubated with rRBCs, many of the pairs were able to form hetero-oligomers with wild-type alpha-hemolysin (s-alpha HL) and most of these formed homo-oligomers in the absence of s-alpha HL. However, only members of a subset of these pairs were able to **lyse** the cells. The lytic combinations contained overlaps, nicks, or gaps, but only two pairs, with nicks between **amino acid** residues 128 and 129 and between 131 and 132 had hemolytic activities approaching that of the wild-type polypeptide. Active combinations could also be obtained by separately translating NH2- and COOH-terminal truncation mutants and then combining them. These findings suggest that the integrity of the central loop is of little significance for oligomer formation but that it is more important for the final step in pore assembly or alternatively for determining the correct structure of the conductive channel. Our findings disagree with previous reports of NH2- and COOH-terminal fragments with hemolytic activity and of the prevention of hemolysis by proteolytic cleavage in the central loop. This discord is attributed to experimental and interpretative ambiguities in the earlier protein chemistry. For example, we show that loss of hemolytic activity after treatment with trypsin is not due to cleavage after Lys-131, as previously proposed, but to the removal of a small NH2-terminal peptide through cleavage after Lys-8.

L5 ANSWER 19 OF 21 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 91349596 MEDLINE
DOCUMENT NUMBER: 91349596 PubMed ID: 1880421
TITLE: Mouse NKR-P1. A family of genes selectively coexpressed in adherent lymphokine-activated killer cells.
AUTHOR: Giorda R; Trucco M
CORPORATE SOURCE: Department of Pediatrics, University of Pittsburgh, School of Medicine, PA.
CONTRACT NUMBER: AI23963 (NIAID)
AI26364 (NIAID)
CA44977 (NCI)
+
SOURCE: JOURNAL OF IMMUNOLOGY, (1991 Sep 1) 147 (5) 1701-8.
Journal code: IFB; 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
OTHER SOURCE: GENBANK-M64137; GENBANK-M64138; GENBANK-M64139; GENBANK-M64140; GENBANK-M64141; GENBANK-M64142; GENBANK-M64143; GENBANK-M77676; GENBANK-M77677; GENBANK-M77678
ENTRY MONTH: 199109
ENTRY DATE: Entered STN: 19911020
Last Updated on STN: 19911020
Entered Medline: 19910927

AB NK cells are a subpopulation of large granular lymphocytes. They are able to recognize and **lyse** a wide variety of virally infected or neoplastic target cells without previous sensitization or MHC restriction. The molecules involved in target recognition and subsequent triggering of the killing process are still undefined. Recently, a 30-kDa protein highly expressed on rat NK cells and capable of mediating **transmembrane** signaling was identified and the gene coding for it cloned and sequenced. To better understand the role of this protein in NK cell-mediated cytotoxicity, we cloned its mouse homologue by cross-hybridization of the rat gene to a cDNA library generated from highly purified mouse lymphokine-activated NK cells. Three messages, differing in size and sequence and encoded by different genes, are specifically cotranscribed in mouse NK cells. The protein products of this gene family express the lectin-like motif characteristic of type II **transmembrane** molecules. Both the rat and mouse proteins have conserved tyrosine and serine residues in their cytoplasmatic portion that are potential

phosphorylation sites. They also share a sequence that could be the binding site of the P56lck tyrosine kinase. These observations are consistent with the signaling function hypothesized for these proteins.

L5 ANSWER 20 OF 21 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 90378305 MEDLINE
DOCUMENT NUMBER: 90378305 PubMed ID: 2399464
TITLE: NKR-P1, a signal transduction molecule on natural killer cells.
AUTHOR: Giorda R; Rudert W A; Vavassori C; Chambers W H; Hiserodt J C; Trucco M
CORPORATE SOURCE: Pittsburgh Cancer Institute, PA.
CONTRACT NUMBER: AI 23963 (NIAID)
AI 26364 (NIAID)
CA 44977 (NCI)
SOURCE: SCIENCE, (1990 Sep 14) 249 (4974) 1298-300.
Journal code: UJ7; 0404511. ISSN: 0036-8075.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-J02762; GENBANK-J02814; GENBANK-K02817; GENBANK-M14766; GENBANK-M62891
ENTRY MONTH: 199010
ENTRY DATE: Entered STN: 19901122
Last Updated on STN: 19970203
Entered Medline: 19901017

AB Natural killer (NK) cells are a subpopulation of large granular lymphocytes characterized by densely staining azurophilic granules. NK cells are able to recognize and **lyse** various virally infected or neoplastic target cells without previous sensitization or major histocompatibility complex restriction. A 60-kD disulfide-linked dimer, highly expressed on NK cells, was found capable of mediating **transmembrane** signaling. The gene encoding this signal transduction molecule was cloned and its nucleotide sequence determined. The encoded protein showed significant homology with a number of lectin-related membrane proteins that share receptor characteristics. This protein may function as a receptor able to selectively trigger NK cell activity.

L5 ANSWER 21 OF 21 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1988:453098 CAPLUS
DOCUMENT NUMBER: 109:53098
TITLE: Induction of human T-cell or natural killer cell with antibodies to T11 glycoprotein, cloning and sequencing of T 11 cDNA
INVENTOR(S): Reinherz, Ellis L.; Siliciano, Robert F.; Sayre, Peter; Chang, Hsiu Ching; Richardson, Neil
PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Inc., USA
SOURCE: Eur. Pat. Appl., 19 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 260880	A2	19880323	EP 1987-308016	19870910
EP 260880	A3	19900307		
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AB A method for turning on the cytolytic effector function of human T-cells or natural killer cells comprises contacting the cells with ligands specific for the T11 sheep erythrocyte-binding glycoprotein of the cells. The cDNA sequence for human T11 is disclosed. The effect of anti-T11 monoclonal antibodies on the lysis of target cells by alloreactive T-cell clones was examd. Anti-T113 in combination with either anti-T111 or anti-T112 induced T-cell clones to **lyse** the lymphoblastoid target Laz509.